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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/785,230	02/25/2004	Tadamitsu Kishimoto	046124-5042-01	1453

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EXAMINER	
GODDARD, LAURA B	
ART UNIT	**PAPER NUMBER**
1642	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/27/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

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Office Action Summary	Application No.	Applicant(s)	
	10/785,230	KISHIMOTO ET AL.	
	Examiner	Art Unit	
	Laura B. Goddard, Ph.D.	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *20 November 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-23 and 25-30* is/are pending in the application.

4a) Of the above claim(s) *1-23,27,29 and 30* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *25,26 and 28* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☒ Certified copies of the priority documents have been received in Application No. 09/646,785.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. The Amendment filed November 20, 2006 in response to the Office Action of May 18, 2006, is acknowledged and has been entered. Previously pending claims 25, 26, and 28 have been amended. Claims 25, 26, and 28 are currently being examined.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office Action.

NEW REJECTIONS

(Necessitated by amendment)

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

3. Claims 25, 26, and 28 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a WRITTEN DESCRIPTION rejection.

The claims are drawn to a method for treating a solid cancer, treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering a substance that inhibits CXCR4 to a mammal in need thereof, wherein the substance inhibits binding between the ligand SDF-1 and the receptor CXCR4, wherein the substance is selected from the group consisting of: i) an anti-CXCR4 antibody, or a fragment thereof possessing binding activity to CXCR4; and ii) an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to SDF-1.

The specification discloses human CXCR4 amino acid sequence SEQ ID NO:1 and murine CXCR4 amino acid sequence SEQ ID NO:3 (p. 14, lines 23-26). The specification discloses human SDF-1-α as SEQ ID NO:5 and its base sequence in SEQ ID NO:6. Human SDF-1-β is amino acid sequence SEQ ID NO:9. Murine SDF-1-α is amino acid sequence SEQ ID NO:7 and its base sequence is SEQ ID NO:8. Murine SDF-1-β is amino acid sequence SEQ ID NO: 10 (p. 15, lines 5-22). The specification does not disclose any other CXCR4's and SDF-1's as broadly encompassed in the claims.

To provide adequate written description and evidence of possession of a claimed genus, the specification must provide sufficient distinguishing identifying characteristics of the genus. The factors to be considered include disclosure of complete or partial structure, physical and/or chemical properties, functional characteristics, structure/function correlation, methods of making the claimed product, or any combination thereof. In this case, the only factor present in the claim is a recitation of "the substance inhibits binding between the ligand SDF-1 and the receptor CXCR4",

"anti-CXCR4 antibody, or a fragment thereof possessing binding activity to CXCR4", and "an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to SDF-1". Accordingly, in the absence of sufficient recitation of distinguishing identifying characteristics, the specification does not provide adequate written description of the claimed genus.

Although drawn to DNA arts, the findings in University of California v. Eli Lilly and Co., 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997) and Enzo Biochem, Inc. V. Gen-Probe Inc. are relevant to the instant claims. The Federal Circuit addressed the application of the written description requirement to DNA-related inventions in University of California v. Eli Lilly and Co., 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997). The court stated that " [a] written description of an invention involving a chemical genus, like a description of a chemical species, 'requires a precise definition, such as by structure, formula, [or] chemical name', of the claimed subject matter sufficient to distinguish it from other materials. " Id. At 1567, 43 USPQ2d at 1405. The court also stated that:

> a generic statement such as "vertebrate insulin cDNA" or "mammalian insulin cDNA" without more, is not an adequate written description of the genus because it does not distinguish the genus from others, except by function. It does not specifically define any of the genes that fall within its definition. It does not define any structural features commonly possessed by members of the genus that distinguish them from others. One skilled in the art therefore cannot, as one can do with a fully described genus, visualize or recognize the identity of the members of the genus. A definition by function, as we have previously indicated, does not suffice to define the genus because it is only an indication of what the gene does, rather than what it is.

Id. At 1568, 43 USPQ2d at 1406. The court concluded that "naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material." Id.

Finally, the court addressed the manner by which a genus of cDNAs might be described. "A description of a genus of cDNAs may be achieved by means of a recitation of a representative number of cDNAs, defined by nucleotide sequence, falling within the scope of the genus or of a recitation of structural features common to the members of the genus, which features constitute a substantial portion of the genus." Id.

The Federal Circuit has recently clarified that a DNA molecule can be adequately described without disclosing its complete structure. See Enzo Biochem, Inc. V. Gen-Probe Inc., 296 F.3d 1316, 63 USPQ2d 1609 (Fed. Cir. 2002). The Enzo court adopted the standard that "the written description requirement can be met by show[ing] that an invention is complete by disclosure of sufficiently detailed, relevant identifying characteristics ....i.e., complete or partial structure, other physical and/or chemical properties, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics." Id. At 1324, 63 USPQ2d at 1613 (emphasis omitted, bracketed material in original).

The inventions at issue in Lilly and Enzo were DNA constructs per se, the holdings of those cases are also applicable to claims such as those at issue here. A disclosure that does not adequately describe a product itself logically cannot adequately describe a method of using that product.

Thus, the instant specification may provide an adequate written description of substances that inhibit binding between the ligand SDF-1 and the receptor CXCR4", "anti-CXCR4 antibody, or a fragment thereof possessing binding activity to CXCR4", and "an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to SDF-1, per Lilly by structurally describing representative antibodies that bind CXCR4 or SDF-1 and inhibit binding between SDF-1 and CXCR4, or describing representative CXCR4 or SDF-1 structures that would produce said antibodies, or by describing "structural features common to the members of the genus, which features constitute a substantial portion of the genus." Alternatively, per Enzo, the specification can show that the claimed invention is complete "by disclosure of sufficiently detailed, relevant identifying characteristics, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics."

In this case, the specification does not directly describe antibodies that bind CXCR4 or SDF-1 and inhibit binding between SDF-1 and CXCR4 useful in the claimed invention in a manner that satisfies either the Lilly or Enzo standards. Although the specification discloses human and murine sequences SEQ ID NOs: 1 and 3 for CXCR4 and SEQ ID NOs: 5-9 for SDF-1, this does not provide a description of the broadly claimed antibodies that bind CXCR4 or SDF-1 and inhibit binding between SDF-1 and CXCR4 that would satisfy the standard set out in Enzo because the specification provides only functional characteristics of the antibodies coupled to inadequately described structural features for CXCR4 and SDF-1 to which the antibody binds.

Further, the specification also fails to describe antibodies that bind CXCR4 or SDF-1 and inhibit binding between SDF-1 and CXCR4 by the test set out in Lilly because the specification describes only human and murine sequences SEQ ID NOs: 1 and 3 for CXCR4 and SEQ ID NOs: 5-9 for SDF-1. Therefore it necessarily fails to describe a representative number of such species.

Thus, the specification does not provide an adequate written description of antibodies that bind CXCR4 or SDF-1 and inhibit binding between SDF-1 and CXCR4 that is required to practice the claimed invention. Since the specification fails to adequately describe the product to which the claimed method uses, it also fails to adequately describe the method.

Further, the following teaching of the court as set out in Noelle also clearly applies to the instant claimed invention. The court teaches as follows: "Noelle did not provide sufficient support for the claims to the human CD40CR antibody in his '480 application because Noelle failed to disclose the structural elements of human CD40CR antibody or antigen in his earlier '799 application. Noelle argues that because antibodies are defined by their binding affinity to antigens, not their physical structure, he sufficiently described human CD40CR antibody by stating that it binds to human CD40CR antigen. Noelle cites Enzo Biochem II for this proposition. This argument fails, however, because Noelle did not sufficiently describe the human CD40CR antigen at the time of the filing of the '799 patent application. In fact, Noelle only described the mouse antigen when he claimed the mouse, human, and genus forms of CD40CR antibodies by citing to the ATCC number of the hybridoma secreting the mouse

CD40CR antibody. If Noelle had sufficiently described the human form of CD40CR antigen, he could have claimed its antibody by simply stating its binding affinity for the "fully characterized" antigen. Noelle did not describe human CD40CR antigen. Therefore, Noelle attempted to define an unknown by its binding affinity to another unknown. As a result, Noelle's claims to human forms of CD40CR antibody found in his '480 application cannot gain the benefit of the earlier filing date of his '799 patent application. Moreover, Noelle cannot claim the genus form of CD40CR antibody by simply describing mouse CD40CR antigen". *Randolph J. Noelle v Seth Lederman, Leonard Chess and Michael J. Yellin* (CAFC, 02-1187, 1/20/2004).

4. Claim 25, 26, and 28 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for **a method for treating a solid cancer, treating a disease pathologically caused by neovascularization, or suppressing vascularization in a mammalian species comprising administering to said mammalian species a substance that inhibits binding between the ligand SDF-1 and the receptor CXCR4 expressed in said mammalian species, wherein the substance is selected from the group consisting of: i) an anti-CXCR4 antibody, or a fragment thereof possessing binding activity to the CXCR4 expressed in said mammalian species; and ii) an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to the SDF-1 expressed in said mammalian species,** does not reasonably provide enablement for a method for treating a solid cancer,

treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering a substance that inhibits CXCR4 to a mammal in need thereof, wherein the substance inhibits binding between the ligand SDF-1 and the receptor CXCR4, wherein the substance is selected from the group consisting of: i) an anti-CXCR4 antibody, or a fragment thereof possessing binding activity to CXCR4; and ii) an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to SDF-1. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to practice the invention commensurate in scope with these claims.

The factors to be considered in determining whether undue experimentation is required are summarized In re Wands 858 F.2d 731, 8 USPQ2nd 1400 (Fed. Cir, 1988). The court in Wands states: "Enablement is not precluded by the necessity for some experimentation such as routine screening. However, experimentation needed to practice the invention must not be undue experimentation. The key word is 'undue,' not 'experimentation.' " (Wands, 8 USPQ2d 1404). Clearly, enablement of a claimed invention cannot be predicated on the basis of quantity of experimentation required to make or use the invention. "Whether undue experimentation is needed is not a single, simple factual determination, but rather is a conclusion reached by weighing many factual considerations." (Wands, 8 USPQ2d 1404). The factors to be considered in determining whether undue experimentation is required include: (1) the quantity of experimentation necessary, (2) the amount or direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state

of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims.

The claims are drawn to a method for treating a solid cancer, treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering a substance that inhibits CXCR4 to a mammal in need thereof, wherein the substance inhibits binding between the ligand SDF-1 and the receptor CXCR4, wherein the substance is selected from the group consisting of: i) an anti-CXCR4 antibody, or a fragment thereof possessing binding activity to CXCR4; and ii) an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to SDF-1.

The claims are broadly drawn to **antibodies that bind to any CXCR4 or SDF-1** protein **from any species**.

The specification discloses human CXCR4 amino acid sequence SEQ ID NO:1 and murine CXCR4 amino acid sequence SEQ ID NO:3 (p. 14, lines 23-26). The specification discloses human SDF-1-α as SEQ ID NO:5 and its base sequence in SEQ ID NO:6. Human SDF-1-β is amino acid sequence SEQ ID NO:9. Murine SDF-1-α is amino acid sequence SEQ ID NO:7 and its base sequence is SEQ ID NO:8. Murine SDF-1-β is amino acid sequence SEQ ID NO: 10 (p. 15, lines 5-22).

The art teaches that CXCR4 and SDF-1 are proteins found in several unrelated species, including non-mammalian species, wherein the proteins do not share 100% or significant homology. For example Blast data for chemokine receptor CXCR4 (pages 1-6 from Signaling Gateway) teach sequence discrepancies and the percent homologies differ between CXCR4 proteins among various species including mouse, rat, human,

shrew, monkeys, marmosets, pigs, cats, dogs, chickens, frogs, zebra fish, etc. Blast data for SDF-1 protein (pages 1-4 from Signaling Gateway) teach sequence discrepancies and the percent homologies differ between CXCR4 proteins among various species including mouse, dog, cat, human, monkey, cow, chicken, pig, etc. CXCR4 and SDF-1 proteins isolated from unrelated species are also taught by iHOP, Information Hyperlinked over Proteins (see page 1 for each CXCR4 and CXCL12, also known as SDF-1). The claims broadly encompass antibodies that bind to CXCR4 and SDF-1 form all species.

One cannot extrapolate the disclosure of the specification to the scope of the claims because the specification does not provide guidance or examples for treating a solid cancer, treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering a substance that inhibits CXCR4 to a mammal in need thereof, wherein the substance inhibits binding between the ligand SDF-1 and the receptor CXCR4, and wherein the substance is selected from the group consisting of: i) an anti-CXCR4 antibody, or a fragment thereof possessing binding activity to ***any* CXCR4** derived from any species; and ii) an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to ***any* SDF-1** derived from any species.

Given the discrepancy in sequence homology between CXCR4 and SDF-1 proteins among different species, one of skill in the art could not predictably treat a solid cancer, treat a disease pathologically caused by neovascularization, or suppress vascularization comprising administering an antibody that binds to *any* CXCR4 or SDF-1 protein from *any* species, because those of skill in the art recognize that the

antibodies binding to proteins derived from one species would not predictably function to inhibit binding of CXCR4 to SDF-1 from an unrelated species.

The art (Tavor et al, Cancer Research, 2004, 64:2817-2824) teaches the treatment of leukemia stem cells in mice after treatment with antibodies that bind to human CXCR4. Antibodies to human CXCR4 decreased the levels of human acute myelogenous leukemia (AML) cells in the bone marrow, blood, and spleen (abstract). Bertolini et al (Cancer Research, 2002, 62:3106-3112) teach that CXCL12/SDF-1 and its monogamous receptor CXCR4 are involved in trafficking of B cells and hematopoietic progenitors (abstract). Bertolini et al teach a tumor challenge trial wherein antibodies to human CXCR4 treated mice with non-Hodgkin's Lymphoma tumors. Tumor growth was abrogated in the majority of mice treated and was significantly delayed in the remaining group (abstract; Figs 5 and 6). Bertolini et al also teach administering antibodies that bind human SDF-1 or CXCR4 *in vitro* to AML cells wherein each of the antibodies reduced the proliferation of AML cell lines and the survival of primary AML cells. Bertolini et al suggests that survival and proliferation of AML cells are mediated by SDF-1/CXCR4 interactions and autocrine secretion of SDF-1 (p. 2818, col. 2; Fig. 4; p. 2819, col. 2 through p. 2820, col. 1).

The art teaches the treatment of conditions caused by neovascularization using antibodies that bind to human CXCR4 or SDF-1. For example, Butler et al (J of Clinical Investigation, 2005, 115:86-93) teach that intravitreal injection of antibodies that bind human SDF-1 prevented retinal neovascularization in a murine model (abstract; Figure 6). Butler et al suggest that blocking SDF-1 function can prevent neovascularization and

may serve as an important advancement in the treatment of ocular disease such as diabetic retinopathy, and that the intravitreal injection of a blocking antibody to SDF-1 can work to block neovascularization in their acute injury model for up to one month (p. 87, col. 1). Butler et al suggests that SDF-1 may be a key player in angiogenesis and in the progression of proliferative retinopathy and that antibodies that block SDF-1 activity may provide a safe and effective alternative treatment for ischemic diseases such as proliferative diabetic retinopathy and diabetic macular edema (p. 91, col. 2). Sengupta et al (Investigative Ophthalmology & Visual Science, 2005, 46:343-348) teach that injection of mice subretinally with antibodies to SDF-1 significantly reduced the size of choroidal neovascularization (CNV) lesions in the eyes (abstract). Walter et al (Circulation Research, 2005, 97:1142-1151) teach that transplantation of bone marrow cells as well as circulating endothelial progenitor cells (EPC) enhances neovascularization after ischemia and that the chemokine receptor is essential for migration and homing of hematopoeitic cells. Walter et al teach that incubation of endothelial progenitor cells (EPC) from healthy volunteers with neutralizing antibodies to CXCR4 profoundly inhibited vascular endothelial growth factor- and SDSF-1-induced migration as well as EPC-induced angiogenesis in an *ex vivo* assay (abstract). Preincubation of transplanted EPC with CXCR4 antibody reduced EPC incorporation and impaired blood-flow recovery in ischemic hind limbs of nude mice (abstract). Walter et al conclude that CXCR4 receptor signaling profoundly modulates the angiogenic activity and homing capacity of cultured EPC (abstract). Finally, Tachibana et al (Nature, 1998, 393:591-594) teach that SDF-1 and CXCR4 define a new signaling

system for organ vascularization and demonstrate that mice lacking CXCR4 or SDF-1 have defective formation of large vessels supplying the gastrointestinal tract. Further, mice lacking CXCR4 die *in utero* and are defective in vascular development, haematopoiesis and cardiogenesis, like mice lacking SDF-1, indicating that CXCR4 is a primary physiological receptor for SDF-1 (abstract).

Again, one cannot extrapolate the disclosure of the specification to the scope of the claims because the specification does not provide guidance or examples for treating a solid cancer, treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering a substance that inhibits CXCR4 to a mammal in need thereof, wherein the substance inhibits binding between the ligand SDF-1 and the receptor CXCR4, and wherein the substance is selected from the group consisting of: i) an anti-CXCR4 antibody, or a fragment thereof possessing binding activity to ***any* CXCR4** derived from any species; and ii) an anti-SDF-1 antibody, or a fragment thereof possessing binding activity to ***any* SDF-1** derived from any species. The art teaches treating tumors, treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering antibodies that bind to the specific CXCR4 or SDF-1 protein expressed in the animal being treated, wherein the antibody interfered with binding of the ligand SDF-1 to the receptor CXCR4. The art does not enable treating a solid cancer, treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering antibodies that bind to CXCR4 or SDF-1 from unrelated species, because those of skill in the art recognize that the antibodies binding to proteins derived from one species

would not predictably function to inhibit binding of CXCR4 to SDF-1 from an unrelated species, which is broadly encompassed by the claims.

Therefore, in view of the state of the art, the breadth of the claims, lack of guidance in the specification, and the absence of working examples, it would require undue experimentation for one skilled in the art to practice the invention as broadly claimed.

5. All other rejections recited in the Office Action mailed May 18, 2006 are hereby withdrawn.

6. **Conclusion:** No claim is allowed. The closest prior art is US Patent 5,543,503, Chuntharapai et al, filed 6/11/1993, issued 8/6/1996. Chuntharapai et al teach treating inflammatory disorders comprising administering antibodies that bind to PF4AR (also known as IL-8 receptor) (Figs. 4a-c; col. 9, lines 10-42; col. 29 through col. 30). IL-8 receptor is identified by Chuntharapai et al as SEQ ID NO:4, which is 100% identical to SEQ ID NO:1 of the instant application identified as human CXCR4 (see sequence search result #2 in issued patents database: 20070206_074028_us-10-785-230-1.rai.). Chuntharapai et al do not teach or suggest treating solid tumors, treating a disease pathologically caused by neovascularization, or suppressing vascularization comprising administering said antibodies.

7. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 C.F.R. ' 1.136(a).

A SHORTENED STATUTORY PERIOD FOR RESPONSE TO THIS FINAL ACTION IS SET TO EXPIRE THREE MONTHS FROM THE DATE OF THIS ACTION. IN THE EVENT A FIRST RESPONSE IS FILED WITHIN TWO MONTHS OF THE MAILING DATE OF THIS FINAL ACTION AND THE ADVISORY ACTION IS NOT MAILED UNTIL AFTER THE END OF THE THREE-MONTH SHORTENED STATUTORY PERIOD, THEN THE SHORTENED STATUTORY PERIOD WILL EXPIRE ON THE DATE THE ADVISORY ACTION IS MAILED, AND ANY EXTENSION FEE PURSUANT TO 37 C.F.R. ' 1.136(a) WILL BE CALCULATED FROM THE MAILING DATE OF THE ADVISORY ACTION. IN NO EVENT WILL THE STATUTORY PERIOD FOR RESPONSE EXPIRE LATER THAN SIX MONTHS FROM THE DATE OF THIS FINAL ACTION.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Laura B. Goddard, Ph.D. whose telephone number is (571) 272-8788. The examiner can normally be reached on 7:00am-3:30pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Shanon Foley can be reached on 571-272-0898. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Laura B Goddard, Ph.D.
Examiner
Art Unit 1642

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